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THE GENESIS OF THE ELECTROCARDIOGRAM

Certain details in the following description are somewhat speculative. Lack of space precludes adequate qualifications.

Ventricular excitation. The more recent evidence largely supports Lewis's earlier conception of the sequence of ventricular excitation. (a) The excitation wave, coming by way of the bundle branches, is delivered simultaneously to the right and left septal surfaces, though initially a larger area of left septal surface is involved. (b) As the excitation wave penetrates the septum from either side, it is distributed via Purkinje fibers subendocardially to the right and left apexes and to those parts of the free wall bordering upon the septal edges. (c) The wave then continues to spread subendocardially to involve the free ventricular walls. Before this subendocardial spread is complete, the wave has already penetrated the whole thickness of the anterior and apical right ventricular wall. In the meantime all the septal muscle has become active. (d) A moment later, the pulmonary conus probably is reached subendocardially; the larger part of the right ventricular wall has been fully penetrated; and the excitation has begun to reach the epicardial surface in the left apical region. (e) Full activation, i.e., throughout the whole thickness of the wall, last occurs in the lateral and lateral basal wall of the left ventricle and, probably, in the pulmonary conus. The subendocardially delivered excitation waves probably move outward through the free walls in a roughly radial fashion.

Instantaneous electrical axes. In order to understand the electrical consequences of the sequence of activation just described it is necessary both to consider the electrical fields produced by the muscle as it changes from the resting to the active state, and also to visualize accurately the orientations of the ventricular walls within the thorax.[1] As the excitation wave moves outward over a broad front through a ventricular wall, all that part of the body in front of the wave is in the positive field, and that part of the body behind the wave is negative. Neither a resting nor a fully active wall produces electrical effects. Without attempting a precise statement, it may be said that the electromotive force which gives rise to the fields may be defined in terms of an electrical axis or vector, which points from the negative to the positive field through the center of that part of the wall through which the wave is progressing (Bayley). The length of the vector is proportional to the electromotive force.

It is a simple matter to demonstrate the presence of the electrical fields. As Wilson and his colleagues have pointed out, when wires from the three extremities used in electrocardiography are brought together to form a common terminal, the electrical potential of this terminal remains practically at zero throughout the cardiac cycle. Hence, when the common terminal and an exploring electrode are connected to the galvanometer, the potential changes recorded are essentially those of the skin surface upon which the exploring electrode rests. If the exploring electrode is so placed that the center of the right ventricle is subjacent, then, after a small, initial R wave, the QRS complex consists of a deep S wave. But when the left ventricle is subjacent to the exploring electrode, the chief QRS deflection is a high R wave. The S and the R, respectively, demonstrate the presence of negative and of positive electric fields, produced mainly by the excitation wave moving outward through the free, left ventricular wall. The vector is now unopposed by a right ventricular vector, since the larger part of that ventricle is fully active. The vector present, for example, at the time instant of inscription of the R apex and tip of the S, is an instantaneous axis.

Spatial vectors. The instantaneous vector just described points in three-dimensional space approximately from the center of the free wall of the right ventricle out through the center of the free left ventricular wall. In an average heart position, this direction is downward, backward, and to the left. The spatial vector is not parallel to the frontal plane, and its effect upon the limb leads depends upon its magnitude and direction as it is projected onto the frontal plane.

The rotation of the instantaneous axes. The QRS complex consists of a succession of instantaneous axes of which the one described above is simply the longest. At the time of the Q wave, which is probably caused by septal excitation, the short vectors point from left to right ventricle at right angles to the upper, anterior part of the septum. The later instantaneous axes, produced by excitation of the free walls, at first point in a roughly base to apex direction; then, while growing in length, they are directed more and more toward the left ventricle. The direction of the longest vector has been given. The later vectors progressively decrease in length and have a progressively more basal direction. The tips of this succession of spatial vectors, as projected onto the frontal plane, describe the QRS loop of the vectorcardiogram.[2]

The mean QRS axis. The mean spatial QRS vector is derived from the directions and magnitudes of the whole series of instantaneous vectors. As projected onto the frontal plane, the mean spatial axis gives the mean QRS axis.

Effects of heart rotation. It is obvious, different persons being compared, that the ventricles may be more or less rotated in either of two directions about three different axes, namely, a transverse axis, a longitudinal axis, and an antero-posterior axis. Hitherto rotations about the latter axis have received most attention, since these are easiest to observe by X-ray, but the effects of rotation about

the other axes are no less important. A common position of the heart in the chest of a sthenic individual may be regarded as one of no rotation about any anatomical axis. The limb lead electrocardiogram may then reveal a Q, at least as a trace, in all leads; an R in all leads, which is not very low in any; and usually a small S in all leads. The peaks of the R waves are synchronous, so that the sum of the heights of R_1 and R_3 equals R_2. If such a heart be rotated about an A-P axis to a somewhat horizontal position, so that the long axis is about at right angles to the line of lead III, R_1 will increase in height and the QRS complex usually becomes "vibratory" in lead III, the individual deflections being very low. If the same heart, unrotated about longitudinal and transverse axes, be rotated to a relatively vertical position, Q_1 becomes less conspicuous or disappears, and S waves are likely to be present in all leads, S_3 being deepest, and R_1 is low.

Restore the heart to its original position of no rotation and then rotate it clockwise on its longitudinal axis, as viewed from the apex. At once R_1 becomes less high, and S_1, if present, deeper. Q_3, if present, becomes deeper, and R_3 is higher. We may pause to consider the reasons. This rotation has directed the longer instantaneous spatial QRS axes backward and almost straight downward in three dimensional space. R_1 is now produced by the earlier apical vectors and S_1 by the late vectors which now project downward, backward, and slightly to the right. R_3 is higher because the large vectors are directed downward, making the left leg more positive than it was before the rotation and making the left arm negative. The sum R_1 plus R_3 no longer equals R_2, since the peaks are not synchronous. The peak of R_1 is earlier than the peak of R_3. If, instead of clockwise rotation, the heart be rotated in a counterclockwise fashion, we observe Q_1 and no Q_3; S_3 and no S_1. R_1 is higher than R_3 and the peak of R_1 is later.

We may now consider rotations about the transverse axis. Again, place the heart in the position first described and then tilt the base forward. The Q vector, which pointed forward, slightly upward and to the right, now points forward, or even forward and downward and all Q waves vanish, as Bayley has pointed out. At the same time, the S waves are deepened and are definitely present in all leads, while the R waves become lower. If this heart is made more vertical, as viewed from in front, R_1 becomes lower and S_1 shallower, R_3 becomes higher and S_3 deeper. If the heart is made very horizontal, the base still being tilted forward, R_1 is followed by a relatively deep S_1 and R_3 is preceded by an unusually deep Q.

If the base of the heart were pushed backward, instead of forward, the heart otherwise being in a position of no rotation, all S waves may disappear and Q waves may appear in all limb leads. The R waves become higher in all leads.

The ventricular gradient. As Wilson has pointed out, if the duration of the active state in every muscle fiber was the same, then, neglecting change in cardiac position during systole, the net area of the T wave in each lead would be equal to the net area of the QRS complex, but of opposite sign. Excepting in digitalization, this is not observed. It follows that the duration of activity differs in different parts of the myocardium. Indeed, there must be differences in closely adjoining regions. The net electrical effect of these differences, as projected onto the frontal plane, is Wilson's ventricular gradient; and it is this which produces the T waves normally observed. If it were possible to stimulate all points in the ventricular myocardium simultaneously, then the electrocardiogram of a normal heart in a usual position would show upright T waves in the three leads, but no QRS complexes. The direction of the mean T-wave vector determined from the electrocardiogram would then be the direction of the gradient, and its manifest magnitude (area) would be the gradient magnitude. Practically, the direction and magnitude of the gradient are determinable from the net QRS-T areas of any two of the limb leads.

Like the mean QRS axis, the gradient is the projection onto the frontal plane of a spatial gradient. Like the mean spatial QRS axis, the gradient has a reasonably consistent direction relative to the masses of the ventricular muscle. The spatial gradient, if it were represented by a long arrow to show its direction, would appear to pierce the heart near the upper margin of the right ventricle in the region of the pulmonary conus and to leave the heart about the center of the free wall of the left ventricle. Like the mean spatial QRS axis, it points, as a rule, backward, downward, and to the left, but it points less far backward than the mean spatial QRS axis. It is calculated that the angle between the mean spatial QRS axis and spatial gradient is 30°; and the gradient forms a spatial angle with the longitudinal anatomical axis of roughly 60°. When the heart is rotated, obviously both electrical axes change their directions in the thorax, but the spatial relations between them and the anatomical axis remain practically constant.

Lack of space forbids further description of these relationships, but it may be pointed out that disease, and particularly local ischemia associated with coronary artery disease with or without infarction, commonly changes the direction of the gradient. The spatial relationships described between the axes then no longer hold. In the absence of significant QRS changes, the limb leads, in conjunction with suitable precordial leads, enable one to detect these gradient deviations. Thorough analysis of these changes is one of the problems for the future.

The gradient is decreased in magnitude by digitalis administration, by increase in heart rate, by the standing posture, in myxedema, etc. Fever, thyrotoxicosis, and probably pregnancy increase it. These magnitude changes produce primary T-wave changes. Secondary T-wave changes result from changes in magnitude of the mean QRS vector, when the gradient is unchanged in magnitude or direction.

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REFERENCES

1. Gardberg, M., and Ashman, R. Arch. Int. Med. 72, 210-230, 1943.
2. Wilson, F. N., and Johnston, F. D. Am. Heart Jl. 16, 14, 1938.